

PATENT
Attorney Docket 036870-5073-02

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Divisional Application of: **Holroyd et al.**)
Application No. **Not Assigned**) Art Unit: **Not Assigned**
Filed: **February 6, 2004**) Examiner: **Not Assigned**
For: **Asthma Associated Factors as Targets for**)
Treating Atopic Allergies Including Asthma)
and Related Disorders)

INFORMATION DISCLOSURE STATEMENT UNDER 37 C.F.R. 1.97(b)

Pursuant to 37 C.F.R. 1.56 and 1.97(b), Applicants bring to the attention of the Examiner the documents listed on the attached PTO-1449 forms. This Information Disclosure Statement is being filed, to the best of the undersigned's knowledge, before the mailing date of a first Office Action on the merits for the above-referenced application. Accordingly, Applicants do not believe that a fee is due with the filing of this paper.

The present application is a divisional application of U.S. Application 10/270,595 (filed October 16, 2002 and now allowed), which is a divisional application of U.S. Application 09/623,624 (filed February 13, 2001 and now U.S. Patent 6,576,434), which is a U.S. National Phase Application of International Application PCT/US99/04703 (filed March 3, 1999). Information Disclosure Statements for U.S. Applications 10/270,595 and 09/623,624 were previously filed. A copy of the PTO-1449 form filed with these Information Disclosure Statements is attached. Also attached is a form PTO-1449 listing the references cited by the Examiner during prosecution of these applications. The Examiner's attention is respectfully directed to the art of record in these prior applications and thus, no references are being submitted.

Applicants respectfully request that the Examiner consider the listed documents and evidence that consideration by making appropriate notations on the attached form. This submission does not represent that a search has been made or that no better art exists and does not constitute an admission that each or all of the listed documents are material or constitute prior art. If the Examiner applies any one of the documents as prior art against any claim in the application, and Applicants determine that the cited document does not constitute prior art under United States law, Applicants reserve the right to present to the office the relevant facts and law regarding the appropriate status of such document.

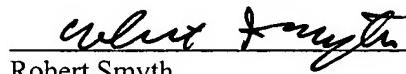
Applicants further reserve the right to take appropriate action to establish the patentability of the disclosed invention over the listed documents, should one or more of the documents be applied against

the claims of the present application.

Except for issue fees payable under 37 C.F.R. 1.18, the Commissioner is hereby authorized by this paper to charge any additional fees during the entire pendency of this application including fees due under 37 C.F.R. 1.16 and 1.17 which may be required, including any required extension of time fees, or credit any overpayment to Deposit Account No. 50-0310. This paragraph is intended to be a **constructive petition for extension of time** in accordance with 37 C.F.R. 1.136(a)(3).

Dated: **February 6, 2004**
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Respectfully submitted,
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INFORMATION DISCLOSURE CITATION (Use several sheets if necessary)			Attorney Docket 036870-5073-02		Application No. Not Assigned		
			Applicant: Kenneth Holroyd <i>et al.</i>		Page 1 of 1		
PTO Form 1449			Filing Date: February 6, 2004		Group Art Unit: Not Assigned		
U.S. PATENT DOCUMENTS							
Initial		Document No.	Date	Name	Class	Sub-Class	Filing Date
	aa	5,733,748	31 March 1998	Yu, <i>et al.</i>	435	70.1	06/06/1995
	ab	6,309,857	30 October 2001	Pauli, <i>et al.</i>	435	69.1	11/17/1998
FOREIGN PATENT DOCUMENTS							
		Document No.	Date	Country	Class	Sub-Class	Translation
	ac	WO 96/39419	12/12/1996	PCT			
OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, etc.)							
	ad	Chu <i>et al.</i> , Glycophorin A interacts with interleukin-2 and inhibits interleukin-2-dependent T-lymphocyte proliferation, <i>Cell. Immunol.</i> 145:223-239 (1992).					
	ae	Cunningham <i>et al.</i> , SA, Cloning of an epithelial chloride channel from bovine trachea, <i>J. Biol. Chem.</i> 270(52):31016-26 (1995).					
	af	Dong <i>et al.</i> , IL-9 induces chemokine expression in lung epithelial cells and baseline airway eosinophilia in transgenic mice, <i>Eur. J. Immunol.</i> (7):2130-9 (1999).					
	ag	Doucet <i>et al.</i> , Interleukin (IL) 4 and IL-13 act on human lung fibroblasts. Implication in asthma, <i>J. Clin. Invest.</i> 101(10):2129-39 (1998).					
	ah	Doull <i>et al.</i> , Allelic association of gene markers on chromosomes 5q and 11q with atopy and bronchial hyperresponsiveness, <i>Am. J. Respir. Crit. Care Med.</i> 153(4 Pt 1):1280-4 (1996).					
	ai	Dugas <i>et al.</i> , Interleukin-9 potentiates the interleukin-4-induced immunoglobulin (IgG, IgM and IgE) production by normal human B lymphocytes, <i>Eur. J. Immunol.</i> 23:1687-1692 (1993).					
	aj	Eklund <i>et al.</i> , Induction by IL-9 and suppression by IL-3 and IL-4 of the levels of chromosome 14-derived transcripts that encode late-expressed mouse mast cell proteases, <i>J. Immunol.</i> 151:4266-4273 (1993).					
	ak	Elble RC, Widom J, Gruber AD, Abdel-Ghany M, Levine R, Goodwin A, Cheng HC, Pauli BU. Cloning and characterization of lung-endothelial cell adhesion molecule-1 suggest it is an endothelial chloride channel. <i>J Biol Chem</i> 1997 Oct 31;272(44):27853-61.					
	al	Eng <i>et al.</i> , Short-term efficacy of ultrasonically nebulized hypertonic saline in cystic fibrosis, <i>Pediatr. Pulmonol.</i> 21:77-83 (1996).					
	am	Kreitman <i>et al.</i> , Site-specific conjugation to interleukin 4 containing mutated cysteine residues produces interleukin 4-toxin conjugates with improved binding and activity, <i>Biochemistry</i> 33(38):11637-44 (1994).					
	an	Levitt <i>et al.</i> , Emerging therapeutic targets in asthma: a role for interleukin-9, <i>Emerg. Thera. Targets</i> , 3:1-11 (1999).					
	ao	McLane <i>et al.</i> , Interleukin-9 promotes allergen-induced eosinophilic inflammation and airway hyperresponsiveness in transgenic mice, <i>Am. J. Respir. Cell Mol. Biol.</i> 19(5):713-20 (1998).					
	ap	Nicolaides <i>et al.</i> , Interleukin 9: a candidate gene for asthma, <i>Proc. Natl. Acad. Sci. USA</i> , 94(24):13175-80 (1997).					
	aq	Petit-Frere <i>et al.</i> , Interleukin-9 potentiates the interleukin-4-induced IgE and IgG1 release from murine B lymphocytes, <i>Immunology</i> , 79:146-151 (1993).					
	ar	Temann <i>et al.</i> , Expression of interleukin 9 in the lungs of transgenic mice causes airway inflammation, mast cell hyperplasia, and bronchial hyperresponsiveness, <i>J. Exp. Med.</i> 188(7):1307-20 (1998).					
	as	Zav'yalov <i>et al.</i> , Nonapeptide corresponding to the sequence 27-35 of the mature human IL-2 efficiently competes with RiL-2 for binding to thymocyte receptors [corrected], <i>Immunol Lett.</i> 31(3):285-8 (1992).					
	at	International Search Report dated July 9, 1999 from International Application No. PCT/US99/04703, 2 pages.					
Examiner				Date Considered			
Examiner: Initial if reference considered, whether or not citation is in conformance with MPEP 609; draw line through citation if not in conformance and not considered. Include copy of this form with next communication to applicant.							

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	aa	6,309,857	10/30/2001	Pauli <i>et al.</i>	435	69.1	11/17/1998
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	ab	Agnel <i>et al.</i> (1999) Identification of three novel members of the calcium-dependent chloride channel (CaCC) family predominantly expressed in the digestive tract and trachea, FEBS Letters 455:295-301					
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